ASTHMA: AN INCREASING PROBLEM AND A CHALLENGE IN MANAGEMENT

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PREFACE

Asthma has everything. It is a problem of the affluent North rather than the impoverished South; and increasing study reveals it to be a many-faceted problem:

- The incidence of asthma is increasing.
- Susceptibility may have a genetic basis.
- Sensitisation in infancy appears to be environmentally influenced.
- The crucial environmental factors appear to be those of the domestic micro-climate.
- Life-threatening attacks can be triggered at any age.
- Triggers are many, both natural and man-made, and they are not necessarily related to the original cause of sensitivity.
- Knowledge and planning, enabling a timely response to severe attacks, makes many asthma deaths preventable.

More than may be so for most other medical conditions, asthmatics respond to informed and comprehensive management. In the case of asthma, there is a prize to be won.

The Royal Society, in conjunction with the National Asthma Campaign, held an afternoon/evening symposium at the Society's hall on this important topic. The speakers were three medicos and a botanist, and summaries provided by them are included below.

The National Asthma Campaign (NAC) began with a pilot media communication program in 1988 which brought together the major organisations in asthma including the business community. This initial venture led to the launch of the NAC in 1990. It consolidated the shared initiative of the Thoracic Society of Australia and New Zealand, The Royal Australian College of General Practitioners, the Pharmaceutical Society of Australia and the Asthma Foundation and was supported by the pharmaceutical industry.

The goals of the NAC are:

- to achieve correct diagnosis of most people with asthma;
- to bring about a decline in preventable deaths from asthma;
- to establish team management of asthma by patient, doctor and pharmacist, so that most people with asthma will be following an individual action plan, developed with their doctor and assisted by their pharmacist.

MICHAEL ABRAMSON

CAUSES AND TRIGGERS OF ASTHMA

It is not as simple to distinguish between the causes and triggers of asthma as might be expected. As a respiratory physician who only treats adults, I normally see asthmatic patients many years after the initiating events. For this presentation, I propose to discuss *causes*, by which I mean the genetic susceptibility and exposures which usually occur in early childhood, that lead to the development of asthma in the first place. I will then proceed to discuss *trigger factors* which can provoke an attack of asthma in someone who already has the condition. To complicate matters, some exposures are capable of acting both as causes and triggers.

Causes of asthma

A helpful model of the development of asthma is summarised in Fig. 1. Asthma comprises at least two underlying traits: allergy and bronchial hyperreactivity. Allergy is the tendency to produce excessive amounts of a particular type of antibody (IgE) in response to otherwise innocuous foreign proteins (allergens) in the environment. Bronchial hyperreactivity (BHR) is the tendency of the airways to narrow excessively in response to allergens or other nonspecific stimuli. Allergy and BHR are seen in most (but not all) patients with asthma.

Asthma genes. There is no doubt that asthma runs in families and some of the genes responsible for this have recently been discovered. Researchers in Oxford have identified mutations in one of the IgE receptor genes on the long arm of chromosome 11, which they claim are associated with allergies. Other groups have been unable to confirm these observations. We have found linkage between this location on chromosome 11 and BHR, rather than allergies (Van Herwerden et al. 1995). This suggests that other genes on chromosome 11 may be responsible for the susceptibility to asthma and the search for these genes is already underway.

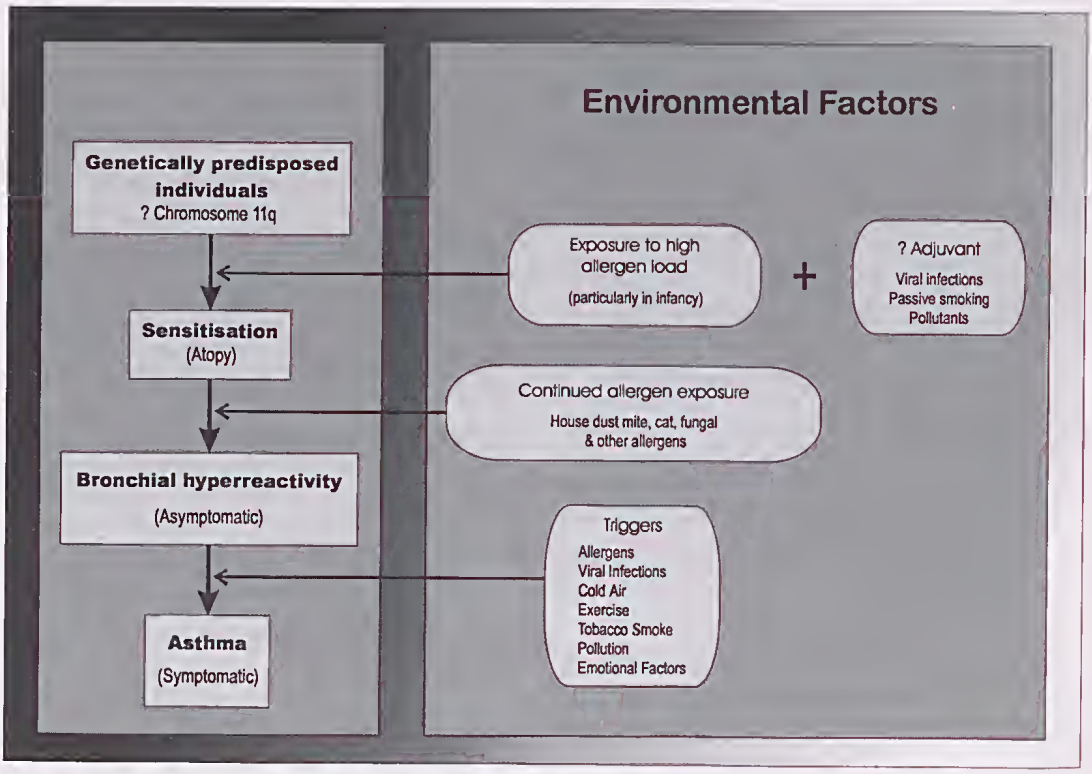

Fig. 1. Hypothesised progression from allergy to BHR to asthma.

There has also been considerable interest in a cluster of genes on the long arm of chromosome 5. Researchers at Johns Hopkins have found evidence for inheritance of high IgE levels in the Amish linked to a location near one particular gene (Interleukin 4) on this chromosome. Researchers at Southampton have found linkage between IgE levels and another gene (Interleukin 9) in this region. Furthermore, an American–Dutch collaboration has linked a nearby location to BHR in asthmatic families from the Netherlands. These exciting developments in the genetics of asthma have been well reviewed elsewhere (Wilkinson et al. 1996).

Childhood exposures. One of the complexities of asthma is that it arises from an interaction between the genes and environmental exposures, mostly occuring in infancy and early childhood. The developing immune systems of young children are bombarded with a wide variety of allergens in Western societies. These include the fine faecal particles of house dust mites, which are inevitable inhabitants of our bedding and carpets. New Zealand researchers have recently found astronomical levels of dust mite allergens in babies' sheepskin bedding. There is good evidence from Southampton that exposure to high levels of dust mites during the first month of life substantially increases the risk of developing dust mite allergy and asthma later in childhood (Warner & Warner 1996).

Pet ownership is common in Australia. Cat allergens are like the smile of the Cheshire cat; they hang around indoors for a long time after the cat has disappeared. Again the Southampton group has found that exposure to cats during the first month of life substantially increases the risk of developing cat allergy and asthma later in childhood. Contrary to popular belief, we now know that milk does *not* make mucus. However there is considerable interest in the role of dietary factors in the development of asthma. Children are now exposed to a wide variety of food additives from an early age. Recently it has been suggested that regular intake of oily fish may lessen the risk of asthma.

Occupational exposures. Although it commonly starts in childhood, asthma can begin at any age. Occupational asthma is a good example of the progression from susceptibility to allergy to BHR and symptoms. The causes can be classified as animal, vegetable, microbial or mineral (Newman-Taylor 1988). Susceptible laboratory workers who handle furry animals develop allergies and asthma in much the same way as children exposed to dust mite or cat allergens. A slightly different process occurs in carpenters exposed to western red cedar sawdust. In this situation, a small molecule needs to bind tissue proteins to set off an allergic reaction, which eventually results in asthma.

There are many other high risk occupations, such as aluminium smelting where occupational asthma has been recognised as a problem for 60 years, but the cause has not yet been identified. One of the key features of occupational asthma is the window of vulnerability. It is during the first few weeks of exposure in a new job that most cases of sensitisation arise. Another important concept is the minimum exposure dose and considerable effort has gone into defining safe exposure limits. Very high exposures to irritant gases such as sulphur dioxide or chlorine will result in a condition similar to occupational asthma, even in workers with no predisposing features.

Triggers of asthma

Some of the causes of asthma can also act as triggers for attacks, once the condition is established. This is certainly the case for house dust mite and cat allergens. On the other hand, air pollution doesn't cause asthma, but can probably trigger attacks. Many asthmatic patients are affected by a wide range of nonspecific irritants including cigarette smoke, perfume, hairspray and household bleach. Viral infections and exercise are common triggers for asthma attacks, particularly in children. The role of dietary factors is controversial.

Allergens. Once sensitisation has occurred, further exposure to allergens will trigger attacks of asthma. Thus we advise our dust mite allergic patients to avoid dusting or vacuuming. It was thought that exposure needed to exceed a threshold level of house dust mite allergen to produce symptoms, but there is now evidence of a dose response relationship at lower levels of exposure. The cartoon book *One hundred and one uses for a dead cat* was written by an asthmatic with severe cat allergy. Pollen allergic asthmatics are prone to dramatic seasonal attacks, which will be further discussed by Professor Knox.

I would like to share with you some fairly recent work on mould allergy. Researchers from the University of Sydney demonstrated that allergy to the mould *Alternaria* is the strongest risk factor for asthma among children in Wagga Wagga.

The strongest risk factor for asthma among young adults in Melbourne now appears to be allergy to the mould *Cladosporium*, the black mould many of you probably have growing in your bathrooms (Abramson et al. 1996). We have found higher levels of *Cladosporium* spores in the home of asthmatics and in the home of those with *Cladosporium* allergy than in the homes of nonallergic controls.

Air pollution. It is a popular misconception that the rising prevalence of asthma must be due to air pollution. In fact the prevalence of asthma is much lower in eastern Germany than in western Germany, whilst air pollution was unquestionably much worse in the east. However I wouldn't want you to conclude that air pollution has nothing to do with asthma. There have been some intriguing experiments suggesting that pre-exposure to ozone or nitrogen dioxide increases the response of the airways to allergens such as house dust mite or grass pollen (see Abramson et al. 1995). Both these gases are major constituents of photochemical smog, which certainly occurs in Sydney and Melbourne, although not to the same extent as in Los Angeles.

In Western societies, indoor air pollution probably results in more potential adverse health effects than outdoor air pollution. Nitrogen dioxide is also given off by unvented gas appliances and can accumulate in quite high concentrations indoors. A recent study in East Anglia found that gas cooking was associated with respiratory symptoms and impaired lung function in women, but not men (Jarvis et al. 1996). Other indoor pollutants which probably trigger asthma include woodsmoke and formaldehyde, which is released by foam insulation in mobile homes.

Cigarette smoking. There was an epidemic of cigarette smoking after World War II, which has only recently been brought under control. Active cigarette smoking was finally proved to cause lung cancer by the US Surgeon General in 1964 and chronic bronchitis and emphysema not long after. There is now a scientific consensus that passive smoking has deleterious effects in asthma (see Abramson et al. 1995). Furthermore, environmental tobacco smoke may act as a cofactor with allergens in the development of allergies in children. These were among the pieces of evidence cited by Justice Morling in his landmark ruling, which has seen smoking banished from most workplaces.

Viral infections. The most frequent triggers of asthma attacks are upper respiratory tract infections (or colds). New highly sensitive techniques have shown evidence of viral infections in over 80% of wheezing illnesses in children (see Abramson et al. 1995). Conversely wheezing has occurred in 50% of respiratory viral infections in asthmatics followed prospectively. Although many studies have suggested an association between early respiratory illness and asthma, it is not at all certain that this is the cause and effect. Indeed there is some evidence that early respiratory infections may protect against the subsequent development of allergies.

Dietary factors. Many patients with asthma report that particular foods worsen their condition. However these reports are notoriously difficult to confirm with placebo controlled double blinded food challenges. The parents of asthmatic children frequently attempt dietary modification, with little objective evidence of benefit. There are well documented reports of asthma attacks following the ingestion of food additives such as tartrazine and metabisulphite. Monosodium glutamate (MSG) which is used as a flavour enhancer in Chinese food, has received bad press as a trigger for asthma, but does not seem to have much effect on lung function tests performed in our laboratory. On the other hand, so-called 'natural' products such as Royal Jelly can cause life threatening allergic reactions in asthmatics.

Exercise. Exercise particularly in cold dry air is a potent trigger of asthma attacks. This is the reason that many asthmatic children have been encouraged to take swimming lessons. Warm moist air is much less likely to trigger attacks. It is thought that cold air dries out the lining of the airways causing the release of chemicals such as histamine, which make the muscles surrounding the airways contract and bring about an attack of asthma. These attacks can be effectively prevented by premedication.

Prospects for prevention of asthma

To prevent asthma developing in the first place will require much more knowledge of the underlying causes. The recent advances in genetics will hopefully allow us to more accurately identify infants at a high risk of developing the condition. The environmental modifications required are likely to include a major redesign of Western style housing. Occupational asthma can be prevented by substitution of safer chemicals and control of

workplace exposures. The prevention of attacks in people who already have asthma will be discussed by Dr Pierce.

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DON CAMPBELL

WHO GETS ASTHMA? WHO DIES FROM IT?

Summary

The prevalence of asthma in Australia has risen in recent years. The prevalence of current asthma in South Australia rose from 5.6% to 9.0% between 1987 and 1990. In 1994 the cumulative prevalence of asthma in the Australian population was 15.3%. Studies of asthma prevalence in 8- to 11-year-olds conducted in the same community demonstrate a rise in prevalence of current asthma from 6.5% to 9.9% between 1982 and 1992, and a rise in the cumulative prevalence of diagnosed asthma from 12.9% to 19.3%.

Genetic factors are important in the development of asthma and atopy, however environmental factors appear to be crucial to the onset and persistence of asthma. These factors include changes in the indoor levels of house dust mite, levels of oxides of nitrogen and possibly diet (oily fish intake). Up to two-thirds of asthmatic children continue to suffer from asthma through puberty and into adult life. Five to 10% of children with mild asthma are likely to develop severe asthma in later life. Children with moderate to severe asthma are at risk of long-term effects of asthma throughout life.

The age-adjusted asthma mortality rate in Australia increased from 2.7/100 000 in 1971 to 4.85 in 1989, declining to 3.53 in 1992. Between 1981 and 1992, for the very young (<15 years) the mortality rate was very low (<1/100 000) with little evidence of a change. For intermediate age ranges there was evidence of an initial rise and a subsequent reduction in mortality rates, whilst in the elderly mortality rates from asthma continue to rise. The positive predictive value of death certification due to asthma is very accurate in the under 55 age group, whilst in the over 64 age group the death certificate is not current enough to be used for epidemiological purposes.

Causative factors to explain the rise in mortality due to asthma may include the increased size of the at-risk population (prevalence), or a change in the severity of asthma. Detailed analyses indicate a large burden of preventable mortality due to asthma which reflects poor management including failure to recognise the severity of asthma, failure to manage asthma appropriately and poor compliance with asthma management plans.

Definition

> 'Asthma is a chronic inflammatory disorder of the airways in which many cells play a role. In susceptible individuals this inflammation causes symptoms which are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. It also causes an associated increase in airway responsiveness to a variety of stimuli.'
>
> *From the international consensus report on diagnosis and treatment of asthma, 1992.*

1. *Prevalance of asthma*

The prevalence of asthma in Australia has risen in recent years. The prevalence of current asthma in South Australia rose from 5.6% to 9.0% between 1987 and 1990. In 1992 the prevalence of current asthma in Victorian adults was shown to be 7%. In 1994 the cumulative prevalence of asthma in the Australian adult population was 15.3%.

Studies of asthma prevalence in 8- to 11-year-olds repeated in the same community demonstrate a rise in prevalence of current asthma from 6.5% to 9.9% between 1982 and 1992, and a rise in the cumulative prevalence of diagnosed asthma from 12.9% to 19.3%. In a study of the prevalence of respiratory symptoms in South Australian pre-school children in 1993 the cumulative prevalence of asthma was 22.5%. This figure compares with the estimated prevalence of 17.1% in primary school age children across Sydney, Melbourne, Brisbane and the Upper Hunter Valley from 1992.

II. *Factors associated with the acquisition of asthma*

Genetic factors are important in the development of asthma and atopy, however environmental factors appear to be crucial to the onset and persistence of asthma.

Up to two-thirds of asthmatic children continue to suffer from asthma through puberty and into adult life. Five to 10% of children with mild asthma are likely to develop severe asthma in later life. Children with moderate to severe asthma are at risk of long-term effects of asthma throughout life.

In 1968 a survey of all 7-year-olds in Tasmania was conducted, 85 855 children born in 1961 were surveyed, being 99% of the population. The cumulative prevalence of asthma was 19.0%.

A history of asthma was significantly associated with:

(i) the child being male (OR 1.56);
(ii) having an atopic background (OR 3.86 for hay fever);
(iii) having a family history of asthma (OR 2.63) this effect being additive on a log scale;
(iv) mother being a smoker (OR 1.26).

The familial aggregation of asthma is consistent with a genetic etiology, but also a shared environment.

As part of the study of the prevalence of respiratory symptoms in South Australian pre-school children, the relationship between prevalence of respiratory symptoms and indoor air quality was examined. Using a logarithmic regression analysis, the following factors were significantly associated with increased risk of asthma:

(i) natural gas stove compared with an electric stove (OR 1.24);
(ii) the use of a non-flued gas heater compared with other heating (OR 1.26).

Parental smoking was significantly associated with increased prevalence rates for bronchitis and ever having wheezed (OR 1.21 and 1.24).

Socio-economic status was not associated with prevalence rates for asthma.

Studies to examine the effect of environmental exposure upon the expression of asthma in children have included:

(i) the study of the effect of allergic sensitisation and climate upon the expression of asthma in children aged 8 to 11 years in seven climatic regions in New South Wales. In all regions the prevalence of current asthma was high (24 to 30%). The prevalence of current asthma was higher in 3 coastal regions where sensitisation to house dust mites was high, and in the far west of the state where sensitisation to *Alternaria* was high, compared with the prevalence in three inland regions where the sensitisation to these allergens was lower;
(ii) a study of school aged children in Sydney where sensitisation to house dust mite was shown to be a particularly important risk factor for both airway hyperreactivity and current asthma in Sydney schoolchildren. Children with large skin test reactions to house dust mite allergen were shown to be the group at greatest risk for morbidity affecting lifestyle.

The author suggests that measures to reduce the level of house dust mite in indoor air quality may have an effect upon the rate of sensitisation to house dust mite and of development of asthma.

In an interesting study of asthma and atopy in South-East Asian immigrants in Melbourne, Leung et al. (1994) found that Asian immigrants and Australian born Asians had a higher rate of atopy than in Australians, and that Asians were twice as likely to react to pollens than Australians and 1.5 times as likely to react to dust mite. Pollen reactors had a 4.8-fold increase risk for development of hay fever, and house dust mite monoreactors a 4.5-fold risk of wheeze or asthma. The prevalence of hay fever in Asian immigrants increased significantly with duration of stay in Australia, a lesser association was seen in duration of stay in Australia for asthma.

III. *The epidemiology of asthma mortality in Australia*

The age adjusted asthma mortality rate in Australia increased from 2.7/100 000 in 1971 to 4.85 in 1989, declining to 3.43 in 1992. Between 1981 and 1992, for the very young (<15 years) the mortality rate was very low (<1/100 000) with little evidence of a change. For intermediate age ranges there

was evidence of an initial rise and a subsequent reduction in mortality rates, whilst in the elderly mortality rates from asthma continue to rise.

The positive predictive value of death certification due to asthma is very accurate in the under 55 age group, whilst in the over 64 years age group the death certificate is not accurate enough to be used for epidemiological purposes.

Where death certificates mentioned asthma but did not code the death as being due to asthma there was an excess of deaths attributable to diseases of the circulatory system and respiratory system, in the latter category chiefly due to chronic obstructive airways disease.

Thus Australian data points to the difficulty of distinguishing between asthma and other causes of chronic obstructive airways disease as potential causes of death in the older age groups.

IV. *What are the features of asthmatics dying of asthma?*

Causative factors to explain the rise in mortality due to asthma may include the increased size of the at-risk population (prevalence), or a change in the severity of asthma. Detailed analyses indicate a large burden of preventable mortality due to asthma which reflects poor management including failure to recognise the severity of asthma, failure to manage asthma appropriately and poor compliance with asthma management plans.

A principal objective of the South Australian Asthma Mortality Study was to test whether cases dying of asthma and asthmatics experiencing near fatal attacks (NFAs) were similar with regard to: their personal and psychiatric characteristics; their asthma histories and asthma severity; features of medical and personal management; circumstances surrounding the fatal or near-fatal episode; and whether the episode could have been prevented.

The study period was from May 1988 to June 1991. Persons dying in South Australia during this period were enrolled in the study if 'asthma', 'asthmatic' or 'status asthmatics' was recorded in Part I or II of the death certificate. The death certificates then were reviewed and if the death *may* have been due to asthma, the certifying doctor was interviewed concerning the circumstances surrounding the death. Interviews were then held with the cases' general practitioners, allied health professionals and household or other close acquaintances.

During the same period, NFA cases presenting to accident and emergency departments of Adelaide teaching hospitals were recruited. To be eligible for entry into the study, cases had to have experienced asthma resulting in either respiratory arrest, or a $PaCO_2$ above 50 mmHg and/or an altered conscious state or inability to speak on presentation to hospital. Interviews of an identical format to those for death cases were held with household or other close acquaintances and general medical practitioners.

This study shows that asthmatics dying from asthma share many important similarities with those asthmatics who survive a near-fatal attack. This applies to: the severity of their asthma; the frequency of asthma symptoms; the impact of asthma on school or work attendance; histories of asthma episodes leading to visits to hospital accident and emergency departments, general hospital admissions and admission to intensive care units; the type of primary health care provider; histories of lung-function testing; use of crisis plans; compliance with prescribed medication; and quality of medical management. Help-seeking behaviour during the fatal or near-fatal episode appeared to be similar and histories of previous psychiatric consultation and levels of psychiatric caseness were also similar.

The asthma death and NFA cases included in the present study were not similar in all respects. NFA cases tended to be younger, to include more males, to be less likely to have concurrent medical conditions, to be more likely to have high levels of denial, to have made fewer doctor visits for asthma in the 12 months prior to the attack, and to have made less use of asthma medications in general.

There were 80 cases in the asthma death group and 154 cases in the NFA group. The death cases were significantly older (mean age 52.3 years, and for the near-fatal attack group 36.1 years). Males comprised 39% of deaths and 57% of the NFA group. These differences were significant.

The levels of asthma severity found in this study were similar to those found in previous studies; 73% of asthma death cases and 65% of NFA cases were classified as having severe asthma, compared with 65% who were assessed as having severe asthma in the Victorian asthma mortality study conducted in 1986.

In the present study, 39% of asthma death cases and 45% of NFA cases were reported as having been admitted to hospital in the preceding 12 months, compared with 39% of cases reported to have been admitted in the preceding 12 month period in both the New Zealand and Victorian asthma mortality studies. A history of admission for asthma in the preceding 12 months has been reported to be the strongest single indicator of

risk for subsequent death from asthma, and is also an indicator of subsequent risk for readmission.

The Victorian asthma mortality study reported that 65% of cases (n = 193) had been provided with an appropriate asthma management plan, compared with 37% of asthma death cases and 49% of NFA cases in the present South Australian asthma mortality study. Only 31% of asthma death cases and 38% of NFA cases were reported as having used a peak flow meter at home, which is not significantly different from the proportion (31%) previously reported amongst asthmatics attending accident and emergency departments in 1992. Only 13 of 163 cases were reported to have had a peak expiratory flow meter in the Victorian asthma mortality study performed in 1986.

Prior medical management was assessed as optimal in only 25% of asthma death and NFA cases combined, with no significant difference between the two groups, on the basis of a consensus opinion of a reviewing committee. The perception of the quality of the medical management was extremely difficult to distinguish, however, from the quality of personal asthma management. Individual self management was assessed as poor for 53% of asthma death cases and 67% of NFA cases. However, when the analysis was restricted to subjects aged less than 60 years, similar levels of poor self management were seen for both groups. Reported compliance with prescribed medication from general practitioner reports was similar for the two groups (69% and 58% respectively).

That 53% of asthma death cases and 45% of NFA cases in the present study were regularly using beta agonists without concomitant preventive medication gives cause for concern. The corresponding figure for the Victorian asthma mortality study was 41%. Regular use of oral corticosteroids were reported for 26% of the asthma death and 16% of the NFA cases, with a figure of 16% also applying in the Victorian asthma mortality study, compared with 42% in the New Zealand study from the early 1980s and 69% in the British study from the late 1970s.

Reportedly, 16% of asthma death cases and 24% of NFA cases had purchased medications (inhaled beta agonists) directly from the pharmacist without a doctor's prescription. Asthmatics who purchase beta agonists directly from the pharmacist have been shown to be less likely to consult with family doctors and also less likely to use prescription-only medication. The high rate of repeat purchase of asthma medication on prescription without further medical consultation, as observed for asthma death (24%) and NFA cases (42%), suggests that an opportunity for regular review and education regarding appropriate asthma treatment is not being utilised for asthmatics who have clearly experienced high levels of prior morbidity due to asthma.

Delay in seeking medical care was a feature of the final event for 56% of asthma deaths and 58% of NFA cases. Delay in receiving medical care was more common in the asthma death group than the NFA group. Amongst those cases where the final event was considered either definitely or probably preventable, delay in actually receiving medical care and less ready access to acute medical care distinguished death cases from NFA cases.

Asthma education will need to provide asthmatics with skills to recognise deteriorating control of asthma, and with appropriate strategies to manage the deterioration. At present, many asthmatics appear just to increase the use of inhaled beta agonist therapy, adopting a 'wait and see' approach hoping the situation will improve. It is of concern that in response to increased asthma symptoms only 20% of asthma death and NFA cases were reported to have increased use of oral corticosteroid medication in the prior 12 months, while over 80% of cases in both groups had increased the use of beta agonists.

Overall, 22% of asthma deaths in the present study were assessed as definitely having preventable factors associated, and 8% of the NFA cases were so assessed, compared with 37% of 126 asthma death cases which were assessed as definitely preventable in the Victorian study. Almost two-thirds (62%) of asthma death cases in the present study were considered definitely or probably preventable, compared with 61% of 271 cases in the New Zealand asthma mortality study, and 86% of 90 cases in the British study. A higher proportion of NFA cases (83%) was assessed as having preventable factors definitely or probably present, possibly reflecting the lower incidence of end-stage asthma in this younger group.

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R. J. Pierce

ASTHMA PREVENTION AND TREATMENT

Asthma is a disease of the airways—the branching system of tubes through which we draw air into the lungs, where exchange of oxygen and carbon

dioxide with the blood and tissues occurs. The fundamental abnormality in asthma is inflammation of the inner lining of the airways with swelling, secretion and narrowing of the lumen and air passage. There is also contraction of the bronchial smooth muscle surrounding the airways with resultant further narrowing of the lumen.

The principles of management of asthma involve firstly the prevention and treatment of airway inflammation and secondly relaxation of the bronchial smooth muscle.

Detailed analysis of asthma deaths has revealed that many result from lack of the knowledge about the condition on the part of many patients, and inadequate treatment strategies. The National Asthma Campaign was formed with the aims of reducing the mortality and morbidity in asthma, maintaining normal airway function and a normal lifestyle for patients with asthma. Constituent bodies are respiratory physicians (Thoracic Society of Australia and New Zealand), family doctors (Royal Australian College of General Practitioners), pharmacists (Pharmaceutical Society of Australia) and the Asthma Foundations of Australia.

The aims of asthma management are to minimise symptoms, to maximise lung function and maintain function at its best at all times, identify trigger factors and prevent the development of permanently abnormal lung function, to reduce the risk of death from an acute attack and to achieve the best quality of life for the person with asthma.

The Six Step Asthma Management Plan outlines these principles.

1. *Assess asthma severity*

Assess lung function when the condition is stable, not during an acute attack.

2. *Achieve best lung function*

Treat with intensive asthma therapy until the 'best' lung function is achieved as shown by measurement of spirometry or PEF.

3. *Maintain best lung function—avoid trigger factors*

Identify and avoid trigger factors and inappropriate medications. Peak flow monitoring can be helpful here.

4. *Maintain best lung function with optimal medication*

Treat with the least number of medications and use the minimum doses necessary.

Ensure the patient understands the difference between 'preventer' medications which combat inflammation and 'reliever' which relax bronchial smooth muscle.

Take active steps to reduce the risk of adverse effects.

5. *Develop an action plan*

Discuss and write a plan for the management of exacerbations.

Detail the increases in medication doses and when and how to gain rapid access to medical care.

6. *Educate and review regularly*

Ensure patients and their families understand the disease, the rationale for their treatment and how to implement their action plan.

Emphasise the need for regular review, even when asthma is well controlled.

The concept of the individual asthmatic person taking an active role in the management of his or her condition as part of a team effort with their local doctor and pharmacist is very important in successful management. The best patient outcome is most likely to be achieved when there is a close working relationship between an interested doctor and an informed patient.

BRUCE KNOX

POLLEN AND THE WEATHER

Pollen grains occur in conifers and flowering plants and carry the male gametes. They are released into the air for fertilisation so that pollen is a natural component of the atmosphere in all seasons, but is especially numerous in spring and early summer in temperate climates. Susceptible humans, breathing the pollen-laden air, develop the symptoms of hay fever and asthma. These allergic diseases are triggered by the presence of certain proteins and glycoproteins in some kinds of pollen that act as allergens. These are environmental proteins or glycoproteins, which interact with the human immune system and trigger the allergic response. Given the dramatic increase in incidence of allergic disease that has occurred during the past 30 years, there has been increasing public concern about allergenic pollen. In this talk, we will examine when, where and in what quantities pollen allergens are present in the aerial environment and how it is affected by weather patterns.

Assessment of the amount of pollen in the air is obtained through use of spore traps, which provide quantitative incidence estimates per cubic metre of air sampled on an hourly to annual basis, as levels of efficiency depending on the size of the particle. A pollen calendar has been produced for Melbourne which shows this information graphically for 22 different types of pollen. Tree pollens predominate in winter: ash, birch, olive and wattle are the most allergenic in humans and, with the exception of wattle, are exotic horticultural species. In spring and early summer, grass pollen predominates (26% of the total pollen catch in Melbourne). Grass pollen is the major source of aeroallergens in the external environment, with about 60% of allergic humans being sensitised to this type of pollen. Weed pollens follow in summer, e.g. wall pellitory, which is now the dominant weedy plant in the CBD of Sydney and abundant in central Melbourne.

The seasonal total pollen counts vary widely from season to season, reflecting the intensity of flowering in any year. For Melbourne, the range is from 2319 to 8217 grains m^{-3} $year^{-1}$, similar to cities in Europe with temperate climates. The key factor regulating the seasonal appearance of grass pollen allergens is average temperature, which is precisely correlated with increasing grass pollen counts. This relationship can be used to predict the intensity of the pollen season. In Melbourne, wind direction is important, with northerly and northwesterly winds carrying high levels of grass pollen.

Rainfall during winter can be used as a predictor for the onset of the grass pollen season. Rain is negatively correlated, since the pollen is washed out of the air. Hay fever sufferers can breathe easy, but not so asthmatics. Rainfall merely changes the total allergen load in the atmosphere, causing the pollen allergens to disperse into the atmosphere aerosol. They exist in two different forms, allergen molecules and allergen-containing fine particles. As grass pollen grains remain living cells for only about one hour after entry into the atmosphere, most grains will be dead. In this condition, they are assumed to behave like a sponge in which allergen molecules diffuse from microchannels in the outer walls from the grains when moistened and become dispersed in the aerosol. Allergen-containing fine particles (starch granules) are produced when grains rupture osmotically during rainfall and are released from the single pore into the atmospheric aerosol. There are approximately 700 of these fine particles in each grain and their size ranges from 0.6 to 2.5 μm. In Melbourne on days following rainfall, a 50-fold increase in the number of particles m^{-3} air has been reported. Allergenic starch granules have been associated with epidemics of thunderstorm asthma during the grass pollen season.

A consequence of the release of allergen molecules into the aerosol is that these molecules are free to interact with other types of fine particles that are associated with air pollution. For example, diesel emitted carbon particles (DECP), each 60 nm in size but forming into fractal aggregates, originate from diesel engine exhaust emissions. Such fine particles have recently been shown to have serious health risks in several American cities with polluted air. These DECPs can become loaded with grass pollen allergen molecules, effectively concentrating the allergens, and suggesting a causal mechanism for the increased levels of asthma in cities during episodes of air pollution. Starch granules are, however, the only class of fine particles that have been tested clinically to show they are capable of eliciting an attack of asthma. There is now a need for quantification of the relative allergenic activities of these various fractions of grass pollen allergens and their contribution to the allergenic activity of the atmospheric aerosol.